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ETHER CONVULSIONS

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THE first references to ether convulsions appear in the *Lancet* and *British Medical Journal* in 1927, and, by a curious coincidence, in issues of the same week; they were by S. R. Wilson and K. B. Pinson respectively. Since then, many articles and letters have appeared, but it must be admitted that there has emerged no definite clue as to aetiology or non-empirical treatment. The various conflicting theories are well-known and the literature needs no exhaustive survey or analysis. A complete summary of the position has appeared recently (Woolmer and Taylor). The only factor common to all cases, with one exception, is the ether. Septic toxæmia almost always is present, and frequently excessive heat, the cases occurring more often during very hot weather. The patients are usually youthful or young adults. The other circumstances are variable in the extreme, and the treatment, in a condition where the real cause is unknown, of necessity is equally uncertain. Further confusion is caused by the fact that cases may have convulsions during one anæsthetic and yet be quite normal during subsequent anæsthesia, and vice-versa, while the same ether may be used in another case with no ill-effects.

No suggestion is completely satisfactory. Since the ether does not seem to contain in itself the toxic or convulsant agent, it is likely that one must look to the secondary changes known to be produced in the *milieu interieur* by ether anæsthesia in general. The principal changes are biochemical and consist in the lowering of the blood CO_2 tension and a tendency towards alkalosis.

The purpose of this article is to suggest that the cause of the fits is possibly connected with a temporary derangement of calcium metabolism such as can occur when the physico-chemical equilibrium of the blood is altered by ether anæs-

thesia. Probably several factors co-operate, including that of idiosyncrasy, or convulsion-diathesis, and the present and past health of the patient.

It must be mentioned, in the first place, that this conception originated in an empirical observation—a case of prolonged ether convulsions that responded dramatically to an intravenous injection of calcium chloride. This was given, without much hope, on the grounds that calcium was "good" for convulsive states. The administration of calcium becomes less empirical when one considers the following factors, all of which operate to depress the ionization of calcium in the blood-stream. They all combine in their tendency to produce an alkalosis.

(1). Ether produces an increased depth of respiration which washes out CO_2, particularly with ethyl-chloride-ether sequence. Clinically, in certain sensitive subjects, hypernoea causes tetany, even when the hypernoea results from physical exercise. Tonic spasms (not ether clonus) are sometimes seen in the induction stage, and there may be laryngeal spasm as in tetany, even though the ether vapour is not in an overwhelming concentration. These spasms are preceded by a gradually increasing hypernoea.

(2). Modern methods of anæsthesia, intubation in particular, tend to lower the alveolar CO_2 tension. Although respiration may be very shallow, it is 100 per cent effective as the dead space is by-passed. It is found experimentally that a very considerable alkalosis develops with the progress of anæsthesia.

(3) Sepsis, particularly of the peritoneal cavity, is often present. This is said to depress the level of the serum calcium, but it is difficult to know whether this is a direct result of the sepsis, or (more likely) a by-effect of the vomiting usually associated with abdominal crises. This may be compared with "gastric tetany" and the biochemical sequelæ of pyloric and high intestinal obstruction.

Younger patients are more subject to these convulsions probably because they have a more labile metabolic balance. Not only are abdominal emergencies more common among them, but they are far more easily upset and prone to vomiting than are elderly patients. Children vomiting from

some simple gastric disorder may show the most extraordinary ketosis, and here again is the unguessable factor of idiosyncrasy, for while one child may be extremely ketotic, another may be relatively unaffected by the same degree of vomiting.

Other factors of importance are the blood changes occurring during mild heat-stroke, aided by the effects of atropine in checking heat and fluid loss. It is also possible that oxygen-lack may play a part, for although the blood may be fully saturated with oxygen, the low CO_2 tension inhibits its dissociation. Nervous tissues are highly sensitive to oxygen-lack, and this may render them unduly susceptible to small changes in their chemical environment. This point is difficult to assess, for while the figures of hæmoglobin dissociation curves clearly emphasize the importance of CO_2, it is doubtful whether a moderate decrease in CO_2 could have such profound effects on function, although the presence of an alkalosis may considerably intensify them.

Unfortunately, simple estimations of the serum calcium are not very informative, since it is the ionized and physiologically active fraction which determines neuro-muscular irritability; tetany often occurs with a normal serum calcium level, and sometimes the total figure may be well above normal.

On the strength of these considerations, the writer always had at hand a syringe and a preparation of calcium, but has never had the occasion to use them. None the less, the treatment indicated has been of success in the one case in which it has been tried. The writer is greatly indebted to Dr. F. F. Cartwright, of King's College Hospital, for his kind permission to quote the details. The case was that of a woman, aged 31, with a ruptured ectopic gestation. The operation was performed in the Trendelenburg position, and after 30 minutes anæsthesia (semi-open ether) typical convulsions developed. Dr. Cartwright, who was aware of the writer's views on the subject, gave an intravenous injection of 10 cubic centimetres of a 10 per cent solution of calcium gluconate (Sandoz) and the convulsions ceased almost immediately. It is interesting to note in this connec-

tion that in pregnancy the serum calcium is progressively lowered, and, in fact, from the point of view of calcium metabolism, pregnancy has been compared with hypoparathyroidism. Calcium gluconate (Sandoz) is recommended as it keeps well (obviously a necessity, considering the rarity of the condition) and, unlike calcium chloride, is non-irritant if there is a small perivenous leakage. The injection should be given fairly slowly, but if a vein cannot be entered, owing to the convulsions, 20 cubic centimetres or more may be given intramuscularly.

The writer can only find one mention in the literature of employment of calcium for ether convulsions (H. J. King). This was a case in which treatment was of no avail, and after some considerable time calcium was injected because of its well-known effect in checking neuro-muscular irritability. After the injection, the patient improved temporarily, but the general condition by now was extremely poor and death followed 58 hours after. It is likely that the hyperpyrexia associated with convulsive states in general, and the tendency to heat-stroke in this state in particular, had produced permanent destruction of cerebral tissue. Woolmer and Taylor have successfully employed evipan, and nembutal was given intravenously in one case by Dickson Wright, after a spinal anæsthetic had produced paralysis up to the costal margin. The effect of evipan and nembutal is probably to cause an overwhelming depression of the neuro-muscular mechanism.

One cannot explain why ether convulsions were unknown before 1926 and any theory can be criticized for this failure. The anæsthetics, operations and patients are all much the same; only the methods have changed. The writer's contribution to the many theories of causation rests on observations on two cases only. It may be that an intravenous injection of any hypertonic solution would check the convulsions; this is a matter for trial and experiment. It is, however, a reasonable and plausible suggestion; it attempts to correlate the many apparently unrelated circumstances of the convulsions; if in any way it illuminates the underlying pathology, then it offers a definite line of rational treatment, as opposed to empiricism and symp-

tomatic treatment; and it seeks to draw attention to a much neglected study—the biochemistry of general anæsthesia.

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THE CAUSE AND PREVENTION OF "SO-CALLED" ETHER CONVULSIONS

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Introduction.

DURING a surgical procedure sudden death without a warning is not unknown. Death preceded by convulsive movements is still considered a rarity deserving of comment. Up to the present time the reason for these fatalities is imperfectly understood, and the suddenness of the onset, the violence of the movements, and the unexpected termination thrust distress and bewilderment upon all concerned. The fatality is all the more inexplicable to surgeons who have seen many patients withstand identical operations and leave the theatre in safety and without incident. There is, however, a certain grim satisfaction in an unsolved mystery.

Foreword.

On May 2nd, 1936, Woolmer and Taylor[1] published in the *Lancet* an excellent paper. The authors describe four cases of this condition named "Ether Convulsions"; they reviewed the literature and gave a definite plan of campaign should such a case occur in any future surgical practice.

Repeated reference is made to this article without apologies for the following three reasons: it is one of the best papers on the subject written; from private enquiry I find that these particular tragedies are increasing and are not as uncommon as previously thought; and, finally, their valuable suggestions appeared one week too late to enable me to prevent, or try to prevent, a tragedy of a similar nature.

Woolmer and Taylor lay stress upon the fact that many

of these cases not infrequently occur when the temperature of the day is excessively hot. They pay tribute to Dickson Wright's[2] hypothesis of hyperthermia and comment that heat stroke may play a large part in the etiology of the condition.

The following is a description of two cases of "so-called" ether convulsions which have recently occurred in my own practice.

Case 1. My first was typical; the hot, thundery day, the stuffy theatre; below stairs the anxious parents of a young child, overfat, but, withal, a good little patient, suffering from a virulent retrocæcal, appendicular infection which in a few hours had led to a gangrenous perforation and spreading peritonitis; the dose of atrophine but no morphia, and a quiet, uneventful induction with gas and oxygen followed by ether. No anxiety was felt concerning the anæsthetic, as he had had previously, and without incident, a similar anæsthetic for a tonsillectomy.

No case of intra-abdominal perforation is easy to anæsthetize; this case was not an exception and the abdomen tended to be incompletely relaxed.

During intra-abdominal operations it has been my custom to note the colour of the intestines and the pulsation of their arteries in order to assist me in the assessment of the patient's condition.

In about 15 minutes after the commencement of the induction in this case, and while separating the retrocæcal appendix, I noticed that the intestines became ischæmic and then quite quickly cyanotic, the veins of the mesentery becoming distended. Very soon came a single twitch of the patient's whole body, to be rapidly followed by stronger and more frequent twitchings but not violent enough to embarrass the completion of the appendicectomy. As the local colour had improved I was not unduly alarmed.

After a brief space the body was quieter, but while the peritoneum and abdominal wall were being sutured the twitchings recurred, and, becoming stronger and more violent, necessitated the rapid completion of the operation.

Some five minutes from the commencement of the spasms, and 20 minutes from the induction, I was able to examine the patient, and in so doing saw that the movements were becoming more convulsive in spite of the fact that for some minutes the ether had been discontinued and CO_2 and oxygen substituted.

The patient was a terrifying spectacle. Previously a white, quiet boy, he was now a purplish grey, the eyes widely open, and the pupils large and glaring. Each deep breath was accompanied by a convulsive twitch of the facial, the ocular, and skeletal muscles, so

that he raised himself from the table to arch like a bow from occiput to heel. Here, pausing for a second, he rested in rigid spasm, to drop suddenly with a convulsive wriggle, quivering his head, his eyes, and whole body for all the world as if he were a terrier shaking a rat. Spasm rhythmically followed spasm, pitiful to behold; one helplessly witnessed his seizures and the stronger muscles overcome the weaker.

Continuation of CO_2 and oxygen made little difference to his condition, though, certainly, after a short period, the attacks appeared to be lessening in violence and frequency. Intravenous calcium was given, not without difficulty owing to the size of the vein and the movements of the patient; if only this injection had been evipan instead of calcium the issue might have been different.

Twenty-five minutes after the onset and 40 minutes after the induction, I noticed the extreme heat of the patient's body to the touch and the oppressiveness of the theatre. The axillary temperature was taken, and read 106°F.; the theatre was immediately aired, but while commencing to sponge down the patient there was a final convulsive movement, and respiration ceased. All further restorative efforts failed.

Interlude.

That night I discussed this case with an anæsthetic colleague. I asked him what I could have done; or, alternatively, what he would have done in a similar emergency. His answer was: "Your grandfather controlled tetanic convulsions with chloroform. Chloroform has been used with success in such a case, but the intravenous injection of a barbiturate such as nembutal, evipan, or pentothal is considered a better method."

My reply was that evipan had been suggested as a possibility, but I had discarded it at a time when I mistook a period of quiet due to muscular exhaustion for a period of recovery; and, secondly, had I used evipan with a fatal result the coroner would have justly expressed his grave doubts as to the suitability of such a drug in the hands of one inexpert with intravenous narcosis at a time when spontaneous recovery was still possible.

It is here one must pause to congratulate James Marr[3] who independently preceded Woolmer and Taylor in the administration of evipan, and by his skilful boldness saved his patient's life. Woolmer and Taylor, in one of their cases, bravely using the same drug, also caused the con-

vulsions to cease, but their patient died some thirty or forty hours later without recovering consciousness. In spite of their success these pioneers do not state why evipan was used, nor do they suggest the method by which this drug obtained its effect, particularly if, as their paper leads one to believe, their patients were deeply under an anæsthetic. Why deepen an already deep anæsthetic?

Some five weeks after my first case my ripening conviction as to the causation of this phenomenon and the rationale of treatment were put to a severe and rigid test.

Case 2. There was admitted under my care a baby two months old, a twin boy, the only pair of twins in a family in which five other brothers had, singly and at convenient intervals, preceded their arrival. In this patient nor in any other member of the family was there a history of epilepsy. The twin brother had gained weight in a satisfactory manner, but this baby, whose birth weight was five pounds eight ounces, was frequently vomiting and not gaining in weight. He looked very ill, pallid, and thin; there was no evidence of pyloric stenosis, but a large, right, congenital hernia was present. With the institution of two-hourly feeds vomiting was not frequent, but in spite of the intensive feeding six ounces only had been gained in five weeks. The hernia was uncontrollable by a truss, and I considered that the hernia was the cause of the loss of weight and that herniotomy was essential.

On the day of the operation the child weighed six pounds six ounces. Here we have an infant, and a very sick infant at that, but no sepsis was present; it was a cool day and there was no thunder except from the anæsthetist, who indignantly refused to anæsthetize such a patient—therefore, there was no anæsthetist. No atropine or morphia was given, and I was forced to perform the operation under local anæsthesia. About 15 c.c. of a 0.75 per cent novocain with one drop of adrenalin was suffused into the operation area in the usual manner. Without waiting the usual ten minutes, as is my practice, the operation was commenced about four minutes after the completion of the anæsthesia, the patient at this time having glucose water out of a bottle. All went well until a very large sac was opened and the cord was being dissected from the posterior wall of the sac, for in a congenital sac the cord is invaginated into its posterior wall. At this point, some ten minutes after the induction of the anæsthesia, the same signs were noticed as in my first case—momentary ischæmia of the intestines followed by venous congestion. To the surprise of the sister, I asked whether the baby's condition was satisfactory. She looked again, and said: "No, he has stopped feeding and does not look at all well." By this time the cord was nearly free, and imme-

diately the first twitch occurred. The operation was stopped and all Spencer Wells removed. The twitches ceased. One bleeding point lay along the ilio-inguinal nerve and had again to be picked up; as the Spencer Wells gripped the ilio-inguinal nerve the twitches at once recurred. The operation was hurriedly completed, but by now the spasms were violent. Here again was the purplish grey colour, the dilatation of the pupils, the facial and ocular movements, the attempt at opisthotonos, the convulsive wriggle, and the rhythmical repetition. To me it was the unbelievable recurrence of a dreadful nightmare, a nightmare softened only by interest and amazement that such a puny infant could make such powerful and spasmodic movements. As in the first case, CO_2 and oxygen proved useless.

Into which vein would the "modern" inject his evipan? I looked at the fontanels and quickly turned to search for "grandfather's" chloroform, ordering the patient back to the ward. Hurriedly following, I reached my minute objective and applied the anæsthetic so vigorously that the convulsions rapidly ceased. So did the respiratory movements. After one or two squeezes of the chest respiration recommenced and without any return of the convulsions.

In the first week this patient gained eight ounces, and in the second nine ounces. He is at present living, and I hope will continue to do so happily ever after, for, typical of the maternal instinct, the good six did not matter provided the prodigal returned.

The circumstances leading to a pitiful tragedy paint such a vivid picture upon the mind that the repetition of the incidents are instantly recognized. This infant's convulsions were not epileptiform, they were not novocain poisoning; they were what has been termed "ether convulsions," and they were produced without the use of any ether and without the use of any anæsthetist. What is more, they were cured by deep narcosis with chloroform.

Previous Theories as to the Causation of Ether Convulsions.

The following are theories which have been advanced in the past.

Idiosyncrasy (Hadfield and Kemp).[4] Congestion of the rolandic area by jugular obstruction (Hewer).[5] CO_2 accumulation (Pinson).[6] Over-oxygenation (Mennell).[7] Sepsis. Hyperthermia (Dickson Wright).[2]

In the evaluation of these main theories advanced as to the causation of ether convulsions, it is important to remember the following points: (1) Not one of these factors is constantly present in all cases; (2) fatalities together with

other alarms and excursions occur commonly to the surgeon and anæsthetist who are youthful in experience; (3) ether may be valueless as an anæsthetic in hot countries owing to its rapid volatilization; (4) a patient with a previously raised temperature might easily raise this temperature several degrees by violent muscular effort in a hot and stuffy theatre; and (5) closed circuit anæsthesia is a difficult anæsthetic to give.

It would appear to be more probable that these previously advanced theories are contributory to, or, alternatively, the consequence of imperfect anæsthesia.

Commentary.

Critical analysis of my own two cases leads me to the conviction that two factors were constant: (1) hurry, (2) trauma, the two combining to procure intense stimulation of the patient and resulting in shock.

Hurry.

Natural anxiety to get a seriously ill patient quickly out of the theatre encouraged me in the first case to continue the operation without giving the anæsthetist time to increase the depth of the anæsthesia when the abdomen was found to be insufficiently relaxed, and led me to proceed in spite of the convulsions. In the second, and for the same reason, the operation was commenced before the local anæsthetic had time to suffuse through the whole operation site.

Trauma.

In each case, at the time of the onset of the convulsions, a hypersensitive area was being surgically interfered with. In the first the peritoneum and the vessels of the mesentery, in the second the peritoneum and structures of the cord, and again, a little later, the ilio-inguinal nerve trunk.

Shock.

The causes of primary shock are still imperfectly understood. The individuality of the patient, his physical state, and the quality and quantity of the stimuli all play their

part. It is true to say that "one man's stimulus may be another man's shock."

Those experienced in the use of local anæsthesia recognize that stimulation of a field of imperfect anæsthesia will give rise not only to an unbelievably widespread distribution of sensation, but also to a definite grading of symptoms according to the particular structures or area stimulated.

Remarkably sensitive are the lining membranes of bone and of cavities, the coats of vessels, and certain sphincters. Those who have suffered real pain would agree that pain can so invade the whole being that even the slamming of a door will produce intense exacerbation.

Must not a serious lesion or long-continued pain facilitate stimuli to reach a wide field of nervous tissues? Those so-called simple manipulations, that sudden dilatation, are they not potentially intensely painful and shock-provoking?

Manifestation of Shock.

How are these "hurts" manifested by the patient? (*a*) by sudden cessation of movement in which the patient gives up the unequal contest, or (*b*) by sudden movement in ardent protest at the insult.

Those convulsive movements described as ether convulsions, are they not a midway house between death and struggling resentment, and their violence indicative of a surgical trauma which has provoked an intense and generalized excitation of the whole nervous system?

The Rationale of Treatment.

Evipan, cholorform, even the continuation of ether, each in their turn, have been recorded as controlling so-called ether convulsions. Is it not more reasonable to suppose that these individual drugs produce their result by a common mechanism in that they so deepen narcosis that all reflexes are subdued and complete rest is given to the outraged nervous system?

The Relation of Surgeon and Anæsthetist.

A patient is placed under the care of an anæsthetist in order to be protected from the painful ministrations of the surgeon. The following statements need contemplation.

Occasionally the innate fear of a fatal issue and the consequent imputation of overdosage quite unconsciously prevent the inexperienced anæsthetist from giving a sufficiently protective anæsthetic to a very sick charge.

Born of a genuine desire for the improvement in the post-operative comfort of the patient, there is a present-day tendency for anæsthetists to attempt to keep patients undergoing painful and shock-producing operations in a state of amnesia rather than in a state of anæthesia. Time only will show where this modern trend will mislead us.

It should, however, appear evident that amnesia and sometimes light anæsthesia is only fair to a patient when adequate and skilled anoci-association is being used as an adjunct.

Let us look to the future extension of valuable methods of anæsthetic research, such as the continuous recording of the blood-pressure and the pulse-rate during surgical procedures, as it is only by this means that these contentions will be proved or disproved.

At the termination of a paper it is customary to express thanks to one's colleagues. On two scores this is gratefully admitted. First, that one has been brought up to believe and continues the attempt to ensure that an operation theatre is not a place of dispute between a surgical House of Lords and an anæsthetic House of Commons, but a meeting-ground where surgeon and anæsthetist work together on equal terms for the benefit of a fellow-sufferer. It is trusted, therefore, that the outspoken remarks in this paper will not be interpreted as derogatory in character. And this leads me to the second in thanking my friend and colleague, who, by refusing to give an anæsthetic, enabled me, I hope, to submit that the surgeon shares equally with his anæsthetist in the responsibility for the causation of these phenomena previously named "ether convulsions".

The Treatment of so-called Ether Convulsions.

Prevention is the ideal. Obviation of shock by the skilful administration of a sufficiently deep anæsthetic and the gentle handling of sensitive tissue. Should convulsions occur there is no time to be lost. The anæsthetist must

acquaint the surgeon in order that he can immediately cease his manipulations and remove all possible instruments that are provocative of stimuli, meanwhile the anæsthetist proceeds to maintain his airway and by the most rapid means in his power renders the patient as deeply anæsthetized as possible. At present evipan appears the quickest and most suitable drug for this purpose.

Conclusion.

There is one point not yet explained. Why should the first and fatal case occur in an only child, but the second—possessing six brothers—survive? It is submitted that the first to come to experience can leave behind a direction-post for the salvation of the second and perhaps for others of his fellow-men.

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SEQUELÆ OF ANÆSTHESIA

By GEOFFREY KAYE, M.D.,

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AT the present time especially, anæsthetists are endeavouring to improve the quality of their work by paying attention, not only to the safety of anæsthetics at operation, but also to the elimination of undesirable sequelæ. In assessing these sequelæ, they must depend upon statistics taken either from their own practice, or from the records of public hospitals. Of the latter, the annual reports issued by the anæsthetic department of the State of Wisconsin General Hospital might well serve as a model. Institutional records suffer, however, from various defects. They are frequently incomplete, and they are in any case compiled by a variety of anæsthetists, whose methods and criteria are not necessarily alike. Private records, on the other hand accumulate relatively slowly and need to be taken in conjunction with the wider picture presented by hospital statistics. A good example of the value of personal records as a foundation for the individual anæsthetist's views is given by a recent publication by Lukis.[2] The writer feels that the publication of personal statistics, if frankly presented and sufficiently "controlled," is desirable as a check upon figures obtained from hospital records.

The present series includes 2,520 administrations of inhalational anæsthetics, personally given or supervised by the writer in the period February 1931 to December 1935. Rather more than half of the administrations were performed in hospital practice, and the rest in private practice. The follow-up of the cases was done in this manner: each patient was visited approximately twenty-four hours after the end of the operation, and further information was secured from the surgeon (or, in hospital practice, the

house surgeon), when next encountered. This system, although inferior to that ruling in hospitals with full-time anæsthetic staffs, was the best that could be devised under the circumstances, and probably led to most of the undesirable sequelæ being recorded.

The cases were distributed as follows:

Anæsthetic	Cases	A-Risk	B-Risk	C-Risk	D-Risk
Ethyl chloride	17	17	—	—	—
Ethyl chloride to open ether	360	267	91	2	—
Open ether	38	21	10	3	4
Open ether with CO_2 ...	141	85	49	7	—
Endopharyngeal ether* ...	394	362	32	—	—
Endotracheal ether† ...	907	788	104	11	4
Chloroform	21	9	9	2	1
Chloroform to open ether	7	5	2	—	—
Nitrous oxide oxygen ...	188	48	50	78	12
Nitrous oxide-oxygen with ether supplement ...	124	23	36	62	3
Endotracheal nitrous oxide-oxygen	85	23	26	22	14
Nitrous oxide-oxygen, nasally, for dentistry ...	124	115	7	2	—
Ethylene-oxygen	57	13	27	13	4
Ethylene-oxygen, with ether supplement ...	21	1	12	8	—
Endotracheal ethylene-oxygen	36	19	15	2	—
Total	2520	1796	470	212	42

* Including administrations through Boyle-Davis gag.

† By "endotracheal inhalation" through Magill tubes, 481.

Fatalities.

There has been no fatality "on the table." One fatal case of pulmonary congestion following entry of vomitus into the lungs, was directly attributed to the anæsthetic, and is described in detail below. Two cases of fatal respiratory sequelæ might perhaps have been averted by different anæsthetic technique, as will be mentioned later. No other post-operative deaths were attributed to the anæsthetic, and no useful purpose would be served by recording such fatali-

ties as those following long-standing intestinal obstruction or operations for the removal of extensive cerebral tumours.

Choice of Anæsthetic in View of the Anæsthetic Risk.

In Australia the "standard" anæsthetic is ether, and the gaseous anæsthetics are reserved in the main for the less good "anæsthetics risks." This is illustrated by the following table:

Anæsthetic employed	Cases	A-Risk	B-Risk	C-Risk	D-Risk
Ether (all techniques	1847	1528=82.2%	288=15%	23=1.3%	8=.43%
Gaseous anæsthetics, except minor dentals	511	127=24.8%	166=32.5%	185=36.2%	33=6.4%

The D-risk cases included many patients for cerebral operations who, although often robust, were included in this class owing to the hazardous nature of the operation.

The influence of the anæsthetic risk in determining the choice of anæsthetic may be better appreciated from the following table:

Risk	Cases	Received Gas anæsthesia	Received ether
A	1655	127= 7.7%	1528= 92.3%
B	454	166= 36.5%	288= 63.4%
C	208	185= 89.0%	23= 11.0%
D	41	33= 80.5%	18= 19.5%

The sicker patients, then, received gas anæsthesia, but ether was used for those patients who could tolerate it with reasonable safety. The choice of anæsthetic seldom erred in respect of safety at the time of operation, but, as will be shown, occasionally did so in respect of subsequent respiratory sequelæ. Gas anæsthesia in good-risk patients usually implied some special indication such as the presence of coryza, of mild diabetes or of renal dysfunction. Ether, when chosen for "C" and "D" -class patients, was usually made necessary by economic considerations. Chloroform

was rarely used, and only when demanded on technical or economic grounds.

Post-Anæsthetic Vomiting.

The incidence of vomiting was investigated in 1,295 cases, by inquiry from both the patient and the nursing staff. The results may be tabulated generally as follows:

Operation	Ether				Nitrous oxide				N_2O + ether				C_2H_4				C_2H_4 ether			
	*	†	‡	§	*	†	‡	§	*	†	‡	§	*	†	‡	§	*	†	‡	§
Abdominal	23	46	11	5	6	5	1	—	24	17	3	3	5	3	1	—	11	3	1	—
General surgical	61	123	29	7	87	40	10	7	18	11	5	4	5	3	—	1	1	1	—	—
Nose and throat	134	436	58	9	29	12	—	—	8	7	1	—	12	8	2	—	2	—	1	—
Total	218	605	98	21	117	57	11	7	50	35	9	7	22	14	3	1	14	4	2	—

Code.—* No vomiting. † Slight. ‡ Moderate. § Severe vomiting. Slight vomiting was taken to mean that the patient was comfortable within three or four hours of awakening from the anæsthetic. Moderate vomiting implied vomiting prolonged until the evening of the day of operation, and severe vomiting anything in excess of this.

The lesser vomiting after gaseous anæsthetics as compared with ether was very apparent in the above figures. Further, "slight vomiting" after gaseous anæsthetics implied less discomfort than "slight vomiting" after ether; in fact, it often meant only a single vomit immediately on terminating the anæsthetic, so that the patient might imagine that he had not vomited at all.

The superiority of gaseous anæsthetics over ether is well illustrated in the following table, in which the above figures are expressed in percentage form. In compiling it, the ethylene cases were combined with the nitrous oxide, as being too few for separate analysis and as being apparently comparable.

Anæsthetic	Cases	No vomiting %	Slight %	Moderate %	Severe %
Ether, all techniques	942	23.1	64.2	10.4	2.2
Gases (summed) ...	353	57.5	31.2	7.1	4.2
Nitrous oxide, without ether supplement	192	60.9	29.7	5.7	3.6

It appeared from these figures that the superiority of gaseous anæsthetics was manifested in the "no vomiting" and "slight vomiting" classes, and that the ratio of moderately-severe or severe vomiting was much the same whether gas or ether was employed. The incidence of vomiting did not seem to be materially enhanced in those cases where ether supplement was employed with gaseous anæsthetics; the addition to the anæsthetic mixture of an ounce or two of ether, spread over an operation perhaps for two or three hours, could exert no very deleterious effect.

An attempt was then made to investigate the incidence of vomiting in terms of the operation performed. Ideally, the data should be tabulated according to the several operations; in the present series the number of operations was too small for this course to be profitable. The data were accordingly grouped as in the following table:

Operation	Cases	No vomiting	slight	moderate	Severe
(a) Ether Cases.		%	%	%	%
Abdominal	85	27.0	54.1	12.9	5.9
General surgical ...	220	27.7	55.9	13.2	3.2
Ear, nose and throat	637	21.0	68.4	9.1	1.4
(b) Gas Cases.					
Abdominal	83	55.4	33.7	7.2	3.6
General surgical ...	188	56.4	29.2	8.0	6.4
Ear, nose and throat	82	62.2	32.9	4.9	—

The figures indicate the great superority of gaseous anæsthetics over ether in each operation-group, so far as the milder degrees of vomiting were concerned. The more severe grades had much the same incidence, independently of the anæsthetic used; a fact which may suggest that certain individuals, for biochemical or physical reasons, are prone to severe vomiting whatever anæsthetic they may receive. Certain such patients were encountered in the series. Gas was used for them, as their previous experiences of ether had been unfortunate. Despite all care preoperatively and at operation, these patients generally suffered from severe after-sickness, less only in degree from that

reported by them as following ether. No reasons could, in general, be assigned for this unpleasant response to anæsthetics. Clinically, the patients seemed to belong to either of two groups: (*a*) a group giving a history of digestive instability, the patient being readily nauseated by certain foods or drugs; (*b*) a group in which the history pointed to possible labyrinthine disturbance, evidenced by car-sickness or train-sickness. Speculation, however, would prove less useful than biochemical investigation of these unfortunate individuals.

Reverting to the table, it will be seen that patients for abdominal operations showed much the same incidence of after-sickness as patients for general surgical operations under the same anæsthetic, a result which differed from that anticipated, as interference with the abdominal contents might be expected to produce much reflex vomiting. Patients habitually vomiting before operation were, of course, excluded from the series, as it was impossible to assess post-anæsthetic sickness in them.

Oto-rhino-larynological cases showed relatively little after-sickness, possibly because they involved no great laceration of tissue. Where ether was employed vomiting (mainly of blood) was usual, but was rarely severe. With gas anæsthesia recovery was rapid and swallowing of blood was minimised, so that the incidence of after-sickness was still less than after ether.

Minor dental cases were excluded from the table, their shortness and absence of tissue-trauma making them not comparable. Major oral surgical cases under endotracheal ether or gas anæsthesia were, however, listed as general surgical procedures. Actually, minor dental cases under ether were almost always followed by vomiting, mainly of blood. When gas anæsthesia was employed, vomiting was exceptional, perhaps because of the rapid awakening with little swallowing of blood. This may be illustrated by the following table:

Of 124 cases receiving short gas anæsthetics (by the nasal route) for dental operations:—

Nitrous oxide-oxygen was used in 123 cases and ethylene-oxygen in one.

Ether supplement was required in 11 cases (9 per cent), but no vomiting ensued.

Of 113 cases requiring no ether supplement, vomiting occurred in three only (2.6 per cent).

As remarked above, the series of cases was too small to render it profitable to analyse the separate operations. Evidence existed, however, to show that certain operations were associated with more consistent after-sickness than others. Ocular tendon-transplantation for strabismus in children showed a high incidence of vomiting, whether due to interference with the ocular musculature or to the mere fact of the children being obliged to awaken from the anæsthetic with their eyes bandaged. Operations for toxic goitre were likewise followed by more than the average incidence of vomiting, but this disturbance may be in part attributable to the routine, intensive, post-operative iodine therapy.

The fact that many patients are nauseated by certain sedative drugs is well known, and many such patients were encountered in the present series. Morphine and the more potent barbiturates, such as sodium amytal and nembutal, were the usual offenders; little or no disturbance was to be attributed to the less potent barbiturates such as barbitone. The writer's experience of paraldehyde as a basal narcotic, rectally administered, has been small; contrary to the usual impression, its use was followed by more than the usual amount of vomiting in those cases in the series in which it was employed.

The suggestion has been advanced that induction of "open" ether anæsthesia with the aid of carbon dioxide is associated with less after-sickness than is the case with the ethyl-chloride-ether "sequence". A small series of cases was available for the investigation of this claim.

Method of induction	Cases	No vomiting	Slight	Moderate	Severe
"Open" ether with CO_2 ...	112	23=20.5%	78=69.6%	8=7.0%	3=2.7%
Ethyl chloride-ether	517	121=23.4%	322=62.3%	64=12.4%	10=1.9%

It seems that, although the number of cases is small, little difference existed in the incidence of after-sickness, whichever method of induction was adopted.

One case was encountered in which post-anæsthetic vomiting was the determining cause of death. The patient, an enfeebled man, aged 76 years, received nitrous oxide-oxygen-ether anæsthesia for single-stage removal of the prostate. Biliary vomiting began in the induction stage and persisted through the operation. Post-operatively, it reached such proportions that no fluid could be retained, and dehydration ensued. Rectal and intravenous administration of fluids were of no avail; the patient entered a state of histamine-like shock, to which he succumbed. The cause of the excessive vomiting is not clear, but it is possible that the operation happened to coincide with the onset of uræmia.

The writer has nothing to add to the accepted measures for averting or treating post-anæsthetic vomiting. He believes the taking of concentrated carbohydrate (cane-sugar, boiled sweets, barley-sugar) on the evening preceding operation to be helpful, although he has no statistics to prove this. Glucose has been found somewhat nauseating, and dextrose is preferable, especially for patients who do not like sweetstuffs. Once vomiting occurs, no remedy has been found generally helpful. The drinking of a drachm of sodium bicarbonate dissolved in a half-pint of water is sometimes useful, even if the mixture be returned by the patient. At other times better results have been given by the sucking of ice, the taking of iced dry ginger-ale or soda water, or of citrus-fruit drinks containing much sugar or dextrose. It is often best to allow the patient to make his own choice from the above measures. Post-anæsthetic vomiting, according to various nursing sisters who have been questioned, is certainly less nowadays than ten years ago, with which impression the writer would agree. The improvement is, perhaps, attributable to the modern trend in anæsthesia, which seeks muscular relaxation sufficient only for the needs of the moment, and spares the patient from prolonged, over-deep narcosis. Instead of the former pre-operative starvation, attention is now given to ensur-

ing an adequate reserve of liver glycogen; further, gas anæsthesia is employed wherever the hepatic functions are under suspicion. At the same time, post-anæsthetic vomiting is by no means controlled. Anæsthetists are apt to take it rather as a matter of course, but it is much dreaded by patients, and would justify the further laboratory and clinical studies required for its control.

Respiratory Sequelæ

The incidence of post-anæsthetic bronchitis and pneumonia in the above series of 2,520 cases is given in the following table. The latter does not include cases of transient exacerbation of frankly existing respiratory infection. No case of lung-abscess was encountered in the series.

Anæsthetic	Administrations	Respiratory Sequelæ
Ethyl chloride	17	—
Ethyl chloride to "open" ether ...	360	1
"Open" ether	38	—
"Open" ether with CO_2 induction	141	—
Endopharyngeal ether	394	1
Endotracheal ether	907	5
Chloroform	21	—
Chloroform to "open" ether ...	7	—
Nitrous oxide-oxygen	188	2
Nitrous oxide-oxygen-ether ...	124	3
Nasal nitrous oxide (dental) ...	124	—
Endotracheal nitrous oxide ...	85	1
Ethylene-oxygen	57	—
Ethylene-oxygen-ether	21	—
Endotracheal ethylene-oxygen ...	36	2
	2520	15*

* Equals 0.59 per cent.

The actual operations followed by respiratory sequelæ are shown in the following table:

Operation	Number performed			Respiratory sequelæ		
	Total	Ether, etc.	Gases	Total	Ether, etc.	Gases
Abdominal.						
Gastric	23	9	14	1	—	1
Intestinal	96	66	30	—	—	—
Gall-bladder	63	29	34	1	—	1
Septic	4	—	4	—	—	—
Various	26	14	12	1	1	—
Gynæcological.						
Pelvic	54	39	15	1	—	1
Vaginal	30	11	19	—	—	—
Emergency	2	—	2	—	—	—
Genito-urinary.						
Bladder	15	4	11	—	—	—
Prostate	20	4	16	—	—	-
Renal	17	9	8	—	—	—
Various	18	12	6	—	—	—
Thoracic.						
Empyæma	8	—	8	—	—	—
Thoracotomy ...	7	1	6	1	—	1
Others	3	1	2	—	—	—
Thyreoid.						
Toxic	44	—	44	—	—	—
Non-toxic	3	1	2	—	—	—
Oto-rhinological.						
Tonsils and adenoids	679	634	45	2	—	2
Nasal sinuses ...	383	352	31	1	1	—
Various	246	212	34	4	3	1
General Surgical.						
Bones and joints ...	85	55	30	1	1	—
Breast	22	8	14	—	—	—
Cerebral	39	13	26	—	—	—
Face and jaws ...	50	44	6	—	—	—
Herniotomy	82	73	9	1	1	—
Various	176	109	67	—	—	—
Dental	325	185	140	1	1	
	2520	1885	635	15	8	7

In investigating the reasons for the 15 cases of bronchitis and pneumonia, climatic factors must be discounted, since three of the cases occurred in spring, seven in summer, five in autumn and none in the winter. The apparent reasons for the sequelæ may be classified as follows:

(*a*) *Faulty Choice of Anæsthetic.* Three cases.

1. *Endopharyngeal ether.* Bronchitic woman for laparotomy. Gas declined on economic grounds; chloroform unsuitable. "Warmed ether" was given. Exacerabation of bronchitis; recovery.

2. *Endotracheal ether.* Bronchiectatic child for antrostomy. Chloroform unsuitable and gas not available in that hospital. Bronchopneumonia; recovery. Subsequently given endotracheal nitrous oxide for another operation without disturbance.

3. *Endotracheal ether.* Woman with acute mastoiditis, high fever and rigors, believed to have lateral sinus thrombosis. Sinus found normal but virulent mastoid infection present. Next day had serous labyrinthitis with temperature 106°F. Overwhelmingly toxic pneumonia supervened, with death on third day. The condition may have been septicæmic from the start, the rigors heralding the onset of pneumonia. The administration of ether, however, must have determined a fatal issue.

(*b*) *Technical Errors.* Four cases.

1. *Endotracheal ethylene.* Asthmatic male patient for tonsillectomy. Catheter unduly small and inadequately packed-off. Probable entry of blood into air passages. Mild bronchopneumonia; recovery.

2. *Endotracheal nitrous oxide.* Old man for laryngofissure. Owing to an oversight, the pharynx was not thoroughly aspirated before withdrawing the endotracheal tube. The first deep breath after removal of the tube led to inhalation of blood into the air-passages. Death from bronchopneumonia 36 hours later.

3. *Endotracheal ether.* Adult, male, confined to bed for three weeks by giddiness due to labyrinthitis complicating mastoid disease. Bronchopneumonia supervened on the third day after operation, with fatal results. The patient might have been saved had the complication been reported earlier and CO_2 therapy initiated.

4. *Open ether.* Child, for open reduction of fractured forearm. Unduly long period of post-operative recumbency in splints. Mild bronchitis; recovery.

(*c*) *Various.* Seven cases.

1. *Nitrous oxide-ether.* Old man for gastrectomy. Costal margins were infiltrated with novocain and were very sore post-operatively. Pneumonia, perhaps atelectatic, supervened, with recovery. Three weeks later, with the onset of colder weather, pneumonia returned;

the patient then suffered from three attacks of angina pectoris and succumbed to the last.

2. *Nitrous oxide-ether.* Young man with infected hydatid of lung and copious, purulent expectoration. Anæsthesia difficult, because mask had to be removed periodically whilst pus was expectorated. Pleurisy and pneumonia developed on the unaffected side on the sixth day after operation, due perhaps to the anæsthetic or perhaps to aspiration from the affected lung. The patient recovered.

3. *Endotracheal ethylene.* A young man, suffering from rheumatic carditis, required tonsillectomy. A mild post-operative pneumonia followed, for which the cause is obscure. No obvious technical error existed to explain it.

4. *Nitrous oxide-ether.* An anæmic woman, suffering from arteriosclerosis and angina pectoris, required hysterectomy. The operation was long and difficult. Pneumonia occurred on the third day, with slow recovery. The cause of it is obscure.

5. *Endotracheal ether.* A woman weighing twenty stone, and suffering from asthma and arteriosclerosis, required operation for incisional herniæ. She had, in recent years, undergone 28 operations, nearly all followed by septic sequelæ. On this occasion, the surgical wound sloughed and bronchopneumonia developed on the third day. Recovery ensued. The pneumonia was, in this case, probably atelectatic in origin.

6. *Endotracheal ether.* Healthy woman for dental extractions. History of pneumonia after a previous anæsthetic. On this occasion, developed mild bronchitis, from which she made an uneventful recovery. The ætiology is obscure.

7. *Nitrous oxide-ether.* A child, gravely ill, suffered from double acute mastoiditis. Pneumonia developed after operation, but, as this was of lobar type, it was probably related to the disease-condition rather than to the anæsthetic.

The fifteenth case of respiratory infection merits separate description, as it was the one case in the series in which the anæsthetic was the directly determining cause of death.

A woman, aged 78 years, suffered from abdominal pain thought to be due to cholecystitis. On the morning of operation she vomited profusely, but this fact was not reported by the nursing staff. Anæsthesia was induced with nitrous oxide-oxygen with a small supplement of ether. When the surgeon palpated the abdomen prior to incision, a tide of bile-stained fluid welled up and flooded the patient, respiration ceasing at once. Resuscitation was affected, but the operation was abandoned, it being thought that the patient's only hope of avoiding pneumonia now lay in having an intact abdominal wall, permitting of coughing. Carbon dioxide therapy was at once initiated. The patient, whose expectoration was bile-stained, soon showed respiratory

embarrassment, and died three hours later from acute congestion of the lungs. The revised diagnosis of cholecystic malignancy and gastric dilatation was confirmed at autopsy, revealing a malignant growth of the gall bladder adhering to the transverse colon and pressing on the duodenum. The lungs were congested and hæmorrhagic.

This fatality, although scarcely to be regretted in many ways, would have been directly preventable had the gastric dilatation been recognised and endotracheal anæsthesia adopted.

Of other possible respiratory sequelæ of anæsthesia, three deserve mention, viz. pulmonary embolism, aggravation of existing phthisis and laryngeal trauma. Of the first, no statistics have been collected by the writer, as the evidence does not point towards any relation between this complication and anæsthetic technique employed.

Where pulmonary tuberculosis is diagnosed, gas anæsthesia is commonly employed. Unfortunately, the condition may not be recognized and ether be given, with harmful results. Two such cases were noted in the above series. In the first, an apparently healthy man presented himself for operation for acute mastoiditis. A month later his condition necessitated bilateral radical mastoidectomy. Four months later it was necessary again to operate, when the condition found indicated tuberculous mastoiditis. Death occurred several months later from tuberculous meningitis. The second patient, also apparently healthy, underwent a dental operation, likewise under endotracheal ether anæsthesia. Post-operative pyrexia led to further investigation, which revealed on X-ray gross bilateral pulmonary consolidation, for which almost no physical signs were apparent. The patient was transferred to a sanatorium. In these two cases, then, a latent tuberculous infection was overlooked, and ether was administered to the patient's detriment. How often the same thing may have happened in other cases in the series it is, of course, impossible to determine.

The incidence of laryngeal trauma after endotracheal anæsthesia has been less carefully worked out in this series than should have been the case. Some measure of sore throat, lasting a few hours only, seems to be a not uncommon sequel, although the patient may not refer to it unless specifically questioned. More serious laryngitis or

tracheitis was occasionally observed. Certain cases could be accounted for by trauma to the pharynx or epiglottis, the parts being contused by the laryngoscope either from haste, carelessness or the difficulty of exposing an unfavourably-placed aditus. Although usually transitory, the resulting sore throat lasted in one or two instances for several days. No case of permanent laryngeal damage has come under the writer's observation. In certain cases, however, laryngitis followed perfectly easy intubations, and no obvious cause for it could be found.

In an attempt to ascertain the frequency of laryngeal sequelæ in the above series, a group of 518 records of endotracheal administrations was analysed. No special inquiry was made as to sore throat or hoarseness; the presence of these would only be mentioned on the record if their severity were such as to lead the patient to complain of them. The results may be set out as follows:

Complication	Endotracheal ether by inhalation using wide-bore rubber tubes. 269 cases.	Endotracheal ether by insufflation through small tubes. 192 cases.	Endotracheal nitrous-oxide or ethylene, by inhalation through wide-bore tubes. 57 cases.
Hoarsness without pain	1	1	—
Definite laryngitis or tracheitis ...	3	1	2
Pharyngeal ulceration	4	—	—

Of the laryngitides, one was clearly due to trauma to the epiglottic region in a case in which exposure of the larynx was practically impossible. The two laryngitides after gas anæsthesia both occurred in cerebral cases; intubation was perfectly easy in one, but the presence of a large tube in the larynx for several hours may have determined the

laryngitis.* The other case was probably due to some measure of trauma. Incidentally, of the 57 endotracheal gas administrations, 22 were for cerebral operations. The other three cases of laryngitis were obscure, as no undue difficulty was met in intubation. The cases of pharyngeal ulceration were due to trauma from the mouth-pack or post-nasal plug, either as a result of over-enthusiastic packing by the surgeon or from the use of dry packing gauze, instead of gauze moistened or lubricated with paraffin.

In summary, the incidence of major respiratory sequelæ of anæsthesia in hospital practice seems to have lessened materially since about the year 1928. The writer cannot advance statistics to prove this, but his impression is shared by ward-sisters, who are generally in a position to know. Previous to 1928 respiratory infection was quite common, and the writer is confident that his personal record before that year, had adequate records been kept, would have proved a bad one. The recent improvement has, in all probability, several causes. Better care is taken to safeguard patients from exposure and freer use is made of gaseous anæsthetics in cases where they seem to be desirable. Endotracheal anæsthesia in appropriate cases has become a commonplace of hospital practice, almost every resident medical officer gaining experience in its administration. Finally, unnecessarily prolonged and deep anæsthesia is in disfavour with both surgeons and anæsthetists, whilst full use is made of post-operative inhalations of carbon dioxide.

Other Undesirable Sequelæ.

(1) *Post-operative psychosis.* The number of cases in the present series was not recorded, but was probably five or six. The onset occurred usually several days after operation, and bore no apparent relationship to the anæsthetic technique. The writer has an impression that permanent psychotic changes are rather frequently encountered after the operation of prostatectomy.

* As a result of a recent unfortunate experience in the same operating theatre, it is quite probable that laryngitis was due in this case to the tube not having been cleansed of carbolic before being handed to the anæsthetist.

One case of post-operative psychosis was fatal, although it actually occurred in 1928, before the period of the present report. A middle-aged woman underwent an operation for vaginal repair and sterilization under "open" ether anæsthesia. The course of the operation was uneventful. Next day an acute sexual psychoneurosis developed, leading to death from exhaustion. Autopsy revealed no pathological lesion. Inquiry into the private history disclosed a tragic disharmony of sexual life, leading to a pre-psychotic state for which the gynæcological operation acted as a trigger-release.

(2) *Sore eye.* One patient in the series suffered from corneal abrasion. He had struggled during the induction stage and either received a splash of ether in the eye or, more probably, was injured by the gauze face-cover. In times past the writer saw several cases of "ether sore eye". Most of them were due, not to entry of ether, but to neglect to see that the lids remained closed during anæsthesia. The exposed cornea was then abraded by the face-cover.

Some Notes on Moot's Index.

Moot's index[3] is one of the best-known tests for anæsthetic tolerance. For some years it has been the standard test at the writer's hospital in guiding junior resident medical officers in assessing circulatory reserve. Recently it has received some criticism from cardiologists, who have stated that the index does not adequately represent the reserve of cardiac patients as shown by clinical and electrocardiagraphic findings.

From the anæsthetist's standpoint the test would have value if it would help in forecasting the degree of circulatory depression likely to occur in a given patient during a particular operation and type of anæsthesia. To work this out would have required a larger series of cases than the writer possessed. Unfortunately, the Moot's index is usually calculated when the patient is seen on the evening before operation and has been frequently omitted from the record when the latter is made up on the following day. In consequence, only 693 complete records were available. It was decided to group these according to the Moot's index and to the

anæsthetic risk, in the hope of being able to say whether, given a certain degree of anæsthetic risk, the presence of an abnormal Moot's index would imply a greater liability to circulatory depression at operation.

The results are set out in the following table.*

Moots' Index	"A"-Risk			"B"-Risk			"C"-Risk			"D"-Risk		
	Degree shock*			Degree shock			Degree shock			Degree shock		
	1st	2nd	3rd	1st	2nd	3rd	1st	2nd	3rd	1st	2nd	3rd
25 or under 75 or over	79	3	—	19	11	—	15	14	2	—	5	2
26–39, 61–74	131	3	—	34	8	1	7	10	—	3	3	—
40–60	215	14	1	57	18	1	22	10	1	2	—	2

* Degree of shock was assessed by McKesson's classification, and represents the greatest circulatory depression observed at any stage of the operation, irrespective of the patient's condition at the end of it.

This table, as it stands, is not particularly informative. The data may be more usefully arranged in terms of the anæsthetic risk, as in the table which follows:

446 *"A"-Risk Patients.*

Moot's index	Incidence per cent	Degree of shock seen in those cases: 1st degree	2nd degree	3rd degree
25 or under, 75 or over ...	82 cases or 18.4%	79=17.7%	3=0.7%	—
26–39, 61–74	134 ,, 30.0%	131=29.4%	3=0.7%	—
40–60 ...	230 ,, 51.5%	215=48.2%	14=3.1%	1=0.2%

We infer, then, that the majority of "A"-risk patients had indices between 40 and 60, as might be expected. The incidence of shock in such patients was small, as one would expect from the healthy condition of the patients and the minor nature of the operations. It is interesting to note, however, that abnormal Moot's indices, below 25 or over 75, were relatively frequent in "A"-risk patients, and did not

seem to imply any special liability to circulatory depression at operation.

It will now be helpful to apply similar analysis to the "B"-risk patients.

149 *"B"-Risk Patients.*

Moot's index	Incidence per cent	Degree of shock seen in those cases: 1st degree	2nd degree	3rd degree
25 or under 75 or over ...	30 cases or 20.1%	19=12.8%	11=7.4%	—
26–39, 61–74	43 ,, 29.0%	34=22.8%	8=5.4%	1=0.7%
40–60 ...	76 ,, 51.0%	57=38.2%	18=12.1%	1=0.7%

Here we find that the incidence of circulatory depression much resembles that encountered in "A"-risk patients, although the occurrence of shock of the second and third degree has relatively increased. The presence of an abnormal Moot's index was frequent, although this did not imply any particular liability to circulatory depression.

We may now apply a similar classification to the "C"-risk cases.

81 *"C"-Risk Patients.*

Moot's index	Incidence per cent	Degree of shock seen in those cases: 1st degree	2nd degree	3rd degree
25 or under 75 or over ...	31 cases or 38.3%	15=18.5%	14=17.3%	2=2.5%
26–39, 61–74	17 ,, 21.0%	7= 8.6%	10=12.3%	—
40–60 ...	33 ,, 40.7%	22=27.1%	10=12.3%	1=1.3%

We see here that the Moot's index still lay most usually between 40 and 60, but that the incidence of abnormal indices had increased relatively to "A"- and "B"-risk patients. Further, an increasing percentage of patients showing an abnormal index suffered from circulatory depression of the second or third degree at operation.

The number of "D"-risk patients was so small as to make a table of questionable value. It is given, however, for what it is worth.

17 D-Risk Patients Moot's Index	Incidence per cent	Degree of Shock seen in those Cases 1st Degree	2nd Degree	3rd Degree
25 or under 75 or over	7 cases or 41.2	—	5=29.4	2=11.8
26–39, 61–74	6 ,, 35.3	3=17.6	3=17.6	—
40–60	4 ,, 23.5	2=11.8	—	2=11.8

In this very small series an abnormal Moot's index was usual, and was in each case followed by more or less severe circulatory depression.

If one may draw conclusions from a series of 693 cases, one would say that an abnormal Moot's index was quite a common finding in patients who were clinically excellent "anæsthetic risks". In them it did not seem to have any serious significance. One is tempted to conclude that Moot's index may be ignored when it is found to be abnormal in clinically healthy patients, but that an abnormal index in a patient who is a "bad risk" on clinical grounds should be regarded seriously, because it seems to imply circulatory inefficiency. In other words, Moot's test can supplement, but not replace, a clinical assessment of the patient's condition. It would be interesting to submit others of the well-known tests for circulatory reserve, such as the breath-holding test or the energy index, to similar investigation.

Conclusion.

The writer has presented an analysis of 2,520 administrations of inhalational anæsthetics, recognizing that his experience in other forms of anæsthesia does not entitle him to hold any positive views in regard to them. The cases that have been presented have been followed up and worked out as fully as possible under the conditions in which the writer works. The series is too small for any dogmatic statements to be based upon it, and the writer has tried not to strain conclusions further than their evidence will

admit. If the results have been in some respects satisfactory, they have in other directions given no cause for pride. They have, however, been reported without reserve, because the frank acceptation of bad results must be the first step in their future prevention. It is the writer's hope that other anæsthetists, of wider experience and with greater facilities, will similarly report their statistics. In this way it should be possible to form a true picture of the incidence of undesirable sequelæ of anæsthesia, and of the most effective means of combating them.

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MODERN TREATMENT BY HYPNOTICS AND BASAL NARCOSIS

From the French of H. WEESE, *Anesth. et Analges.*, February 1936.

THE simple effect of narcotics and of hypnotics is to modify physiological reactions or reactive equilibria. If we are to demonstrate their behaviour as a group and to appreciate their value, we must know the physiology and the pathology of sleep itself. Sleep is a physiological phenomenon in which the routes of afferent and efferent excitability are interrupted and in which the diminished excitability of the autonomic nervous system leads to profound extinction of consciousness, that is, the cessation of relations with the exterior world. Fatigue is the preliminary condition most often necessary for the advent of sleep. The seat of this fatigue is the central nervous system, and consequently the phenomena of sleep are the more defined the more developed is the central nervous system. Man passes one-third of his life in sleep. The normal daily sleep of an adult is from six to eight hours. Its depth varies in a manner which is completely characteristic. It is determined by the intensity of stimulant necessary for awakening. Healthy sleep reaches its maximum depth quickly and becomes lighter after an hour or two. The subjects of nervous disorder never attain really deep sleep.

The joint depth and duration of sleep are designated by Frey its "quantity," and this constitutes a measure of the restoration brought about. After great physical effort as well as after a suitable hypnotic the "quantity" can be doubled or trebled by increased profundity of the sleep without its duration being prolonged. As the excitability of the respiratory centre is diminished during sleep in proportion to its depth the tension of CO_2 in the blood, and consequently in the alveolar air, is increased. Determination of

the depth of sleep by estimating the carbonic acid of the alveolar air gives results analogous to those obtained by ending the sleep by means of a measured stimulant. Physiologically sleep appears under two conditions, fatigue and suppression of stimuli. The former hypotheses, according to which brain as well as muscles became fatigued under the incomplete metabolism, particularly of lactic acid or of carbonic acid, endowed with hypnotic action, as Du Bois maintained from observation of hibernating animals, can no longer be admitted. We have rather to think of products, still unknown, of the disintegration of proteids. The moderate acidosis which exists during sleep is not a cause but a consequence of the lowered excitability. Sleep, however, can come on without corresponding labour and consequently without fatigue. The latter is one of numerous preliminary conditions.

Actually sleep is produced by the fact that there is an internal necessity in the mass of cells whereby phases of rest must be introduced into the activity of the organism. Thus one can affirm that sleep is not a purely passive phenomenon, a reaction with regard to some substance or other, but, on the contrary, it is the consequence of a counter-regulation, of a process of excitation, in short, the expression of an activity. The symptoms of sleep are a clear proof of this. In animals one can recognize, in fact, on the part of the sleeping subject muscular contractions such as the prehensile reflex of birds and the upright posture of the horse. Man presents also muscular contraction of this nature, such as contraction of the pupils, upward deviation of the eyes, and the closure of sphincters. W. R. Hess in recent years has made a searching analysis of the active and passive processes which supervene in the sleeping subject. During sleep motoricity is checked in its relationship with conscious processes, for a series of deep reflexes is extinguished. The arrest of motoricity must consequently be looked on as an autonomous symptom of sleep. Respiration and heart-beat are slowed. The excitability of the respiratory centre is lessened. The potassium and calcium of the blood are lowered. The production of urine is diminished. Digestive functions persist in so far as they

are not dependent on conditioned reflexes, that is to say, allied to consciousness.

We find, consequently, in sleep an important selective diminution of animal activities. Among the vegetative functions few are reduced. The reason is, according to Hess, that the relative incapacity which prevents the sleeping subject from perceiving and appreciating external stimuli can be overcome after a few seconds, or at most a few minutes. This inhibitory power of sleep shows, therefore, a regulated selection with regard to certain organs or functions, and represents, in short, an adaptation to the momentary conditions of sleep. This tends—and here, according to Hess is the essential point—to a positive activity in the interest of the individual, having for its object to favour the processes of cellular restoration. After a refreshing sleep the individual possesses a new potential and new reserves in the widest sense of the word. Nevertheless, one is active during sleep.

The pharmacologist is tempted to consider narcosis as a state of profound sleep during which the processes of repair ought to be strengthened, or at least persist without modification. The contrary is observed: during narcosis these processes are diminished. From this point of view narcosis constitutes an inversion. Sleep and narcosis are, it is true, essentially identical, but the curve of restitution oversteps its optimum when deep sleep passes into narcosis.

The teleological order which exists in sleep shows that some apparatus functions in the course of this phenomenon. As opposed to the sympathetic, the parasympathetic regulates chiefly the economic restorative and protective functions of the organism. W. R. Hess considers that it is this which is the motive power of sleep. The regulation of this can be conceived only in the shape of an organic structure. The approximate localization of this controlling centre has been the natural object of a mass experiment carried out by encephalitis lethargica. It is characteristic of this complaint that, alongside of troubles affecting the visual apparatus, there are observed very severe ones which affect sleep, for example persistent somnolence, sleep inversion, dissociation between cerebral and bodily sleep, and, finally, sleepless-

ness. Economo has consistently found, in the brain of subjects dead of encephalitis, inflammatory alteration of the brain matter at the junction of the diencephalon and the mesencephalon. Where there was persistent sleep the lesion chiefly concerned the caudal portion, and when there was insomnia the anterior portion of this region. Economo has therefore conceived the notion of a sleep regulating centre seated in the cerebral peduncles. Hess has established by experiment the existence of this sleep-regulating centre.

For this purpose he introduced fine electrodes into the brains of cats. Electric stimulation of certain regions enabled him to call out alll the symptoms of going to sleep: the animals yawned, took up the posture of rest, and then fell asleep. Hess has then succeeded in lifting the latch of sleep by an artificial stimulus. The cerebral regions, the stimulation of which allowed of this physiological experience, are situated in the grey nuclei of the base, but in front of those the inflammation of which causes pathological somnolence. One is forced to admit that this regulating centre excites the vagus which is awake during sleep. Hess has been able to bring about the predominance of vagal tone by weakening the tone of its antagonist, the sympathetic. Actually it has been possible to put animals to sleep by injecting into the third ventricle ergotamine, a substance devoid of hypnotic action. Economo and Hess consider that it is from this region of the grey matter that sleep is primarily and directly ordered. These authors do not, nevertheless, believe that this collection of ganglionic cells is alone capable of causing sleep. There are reasons to believe that the regulation of sleep is, in fact, ubiquitous and diffuse. It is by the mediation of the vagus that the regulating centre brings on bodily sleep. An inhibition of the hemispheres, starting from this centre, almost completely suppresses consciousness, and an inhibition of the diencephalon interrupts the routes of stimulation and induces cerebral sleep. Our desire for a causation is, nevertheless, not satisfied by the results of the most recent research. It is now a question of knowing the cause of the stimulus. We have already alluded to the "internal rhythm" independently of which one has to admit

a chemical stimulus of the regulation of sleep, a stimulus which will be the consequence of fatigue.

Piéron in 1913 demonstrated the existence of "ponogenous" substances. Legendre and Piéron were able to prevent a dog from sleeping for several days, and on injecting the blood-serum, cerebral extracts, or the cerebrò-spinal fluid of this animal into the fourth ventricle of normal dogs at rest they were able to bring on somnolence and sleep. Their hypnotoxins apparently possessed the following properties: they are soluble in water, insoluble in alcohol, not dialysible or ultra-filtrable, destroyed by oxygen and by heating to 65°C. Marmesco as well as Demole has been able to induce artificial sleep by injecting small quantities of cholin or of $CaCl_2$ in the neighbourhood of the tuber cinereum, and Clöetta has proved that during sleep there is in this region a slight increase in the calcium content. This enrichment in a specific region shows that it is not the question of a cause but simply of a concomitant local symptom or of a consequence of sleep without which the pathological or therapeutic modifications of the blood-calcium would be accompanied by corresponding symptoms of sleep. Jondeck and Bui have proved that during normal sleep or that due to drugs or illness there is an increase of bromine in the region of the centres of the diencephalon and at the same time diminution of it in the hypophysis. They concluded that in the course of this condition a bromo-hormone passes from the hypophysis to the diencephalon. Pincussen, however, showed that their evidence of bromine was not accurate, and the estimation of this hormone of sleep remains at the least doubtful.

Although we do not yet know the physiological ponogenous substances, we must, nevertheless, admit that the regulation of sleep acts on them and protects the other organs against an invasion by these substances. According to Claperède "we sleep not because we are intoxicated by ponogenous substances but in order to protect ourselves against them." Physiology has not hitherto provided any foundation permitting us to realise in cases of insomnia a substitution therapy. Most fortunately, however, we have succeeded in purely empirical fashion in practising a phar-

macological therapy of sleep. We must, it is true, renounce for the time being the induction of sleep by administration of physiological substances, but we are able to satisfy the second of the conditions necessary for sleep, often the most difficult to realise, by the suppression of stimulation. This success is achieved by attenuating temporarily the senses and central excitability by means of hypnotics and narcotics. There is no question here except of the so-called "indifferent" narcotics, narcotics which, as opposed to certain alkaloids acting in an analogous manner, do not enter into chemical reaction with the cells responsible for hypnosis and narcosis. Many hypotheses have been put forward in the hope of enabling us to capture the mechanisms by which hypnotics and "indifferent" narcotics work, and to enable us to find new substances of this kind.

Among all the theories of narcosis the lipoid theory of Meyer and Overton has remained the most fruitful to the present time. This theory affirms that only substances chemically indifferent, soluble in lipoids, can have narcotic action on the protoplasm. Solution of the narcotic in the cellular lipoids is the principal condition of their specific activity which is shown the more quickly and energetically the richer the cell is in lipoids. The relationship between their lipoid and their aqueous solubility determines the activity of narcotics. Meyer and Overton measured this relationship in an olive oil-water system. Since the triglycerides possess no physiological significance in the nerve cell there has always been justifiable mistrust of the experimental basis of the lipoid theory. This mistrust was strengthened when Traube had shown that it was the surface activity, the adhesion pressure (surface tension) which corresponded with narcotic effect, not the relative solubility. Traube's capillary tube experiments do not reproduce living conditions sufficiently exactly to carry conviction. Traube's theory demands, in opposition to that of Meyer and Overton, a certain degree of independence between the structure of the cell and its sensibility to narcosis. Traube's theory has been developed by Warburg. This author insists still more on the part played by the structure of the cell. He has been able to show the inhibition by narcotics of oxidation at the

surface of cystine, for example. In consequence, he contends that the surface at the level of which enzymes exercise their catalytic activity should be invaded by narcotics. So far as inhibition of oxidation is concerned, he has found a formula which permits, given the constants of absorption and of molecular volume of a narcotic, the foretelling of its activity in oxygen inhibition. Warburg has further shown that with narcotics one can succeed in inhibiting fermentation of yeast.

Warburg's theory has been subjected to criticism by Meyer and Hemnis. They rightly insist on the fact that in the living cell there is no surface so clearly defined as that of charcoal used in experiments. In this the atoms are joined together in a manner to offer resistance to the penetration of the substance much more effectively than can the surfaces, steeped in lipoids, of the proteins and carbohydrates. These authors also demonstrate that the concentration of narcotics capable of inhibiting yeast fermentation coincides very exactly with that which precipitates nucleoproteids. But as this concentration is of an order very much greater than that which can cause narcosis in tadpoles, and as further oxidation of cystine in the presence of carbon is of an order much less than that of the tadpole experiments, Meyer contests that the same phenomenon is concerned in every instance.

Warburg's experiences have shown, it is true, that at the level of surfaces oxidations are inhibited by narcotics according to a definite law. But no experiment yet performed on an animal can show that narcosis results from inhibition of oxidation in Warburg's sense. Still more Schlossmann has shown that tissue exchanges are scarcely inhibited by narcosis. Anoxæmia can certainly induce a condition like enough to narcosis, but the phenomena are quite different. Winterstein, truly, finishes his book on narcosis with the conclusion: "the mechanism of action of narcotics is founded on the fact that these substances are easily absorbed by the structural elements of the living organism." Nevertheless, the hopes which were founded on the theory of absorption have not been realised. Meyer and Henuni have striven anew to consolidate the foundations of the lipoid theory. In their

researches they have directed themselves to the weak point of the lipoid theory, that is to say, to the discovery of a substance which can truly provide a representative of the organic lipoids. As the cerebral lipoids contain, alongside cholesterin which possesses but one hydroxyl group, substances such as cerebrin or kerasin which possess several, they have chosen an indifferent substance, olein-alcohol, in which the HO and HC groups present intermediary relations. By tadpole experiments they succeeded in deducing from the relationship between olein alcohol-water and the narcotic concentration confirmation of the Meyer-Overton theory which, according to them, would be still more exact if one could find a still better representation of the organic lipoids. Traube, in his answer, draws attention to the fact, which he demonstrated by the behaviour of various alcohols, that the lipoid solubility besides other physico-chemical properties of narcotics must play a part sometimes decisive.

A satisfactory theory of narcosis cannot be formulated without taking into account the sum of the physical and chemical properties of narcotics. Not one of the current theories deserves to be called a theory of narcosis, for not one of them permits deep penetration into the nature of the phenomena or represents more than a simple rule, a manner of reducing to a formula a collection of facts. The nature of narcosis has been brought nearer to us by the researches of Vernst, Hober and others on the modifications of permeability. We can put trust in the work of Winterstein. This author has succeeded, using the membrane of muscle, in showing that under the influence of an indifferent narcotic there supervenes a reversible modification in the permeability of the membranes for water and for substances soluble in the water. This phase corresponds to the reversible diminution of excitability which appears during narcosis. Stronger and more poisonous concentrations bring on an irreversible increase of permeability and in consequence definite tissue lesions. The work of Schlominsky and of Adler, who have been able by weak galvanic currents either to institute a true electric narcosis or to deepen narcosis brought about by indifferent narcotics, speaks in the same sense. Look at theories of narcosis how we will, the fact

remains that the concentration of the narcotic in the central nervous system is ruled by that of the blood. The concentration in the blood is the result of rapidity of absorption and of its contrary, detoxication. There results the falling asleep, the sleep of long duration or the narcotic effect according to the relationship between absorption and elimination.

[*To be continued.*]

THE DIPLOMA IN ANÆSTHETICS

THE following are the names of the candidates who were recommended by the Examiners for the Diploma after the May examination:

Beaver, Robert Atwood, B.M., B.Ch. (Oxon), L.R.C.P., M.R.C.S.
Blatchley, Donald, M.B., Ch.B. (Edin.).
Canwarden, Henry, L.R.C.P., M.R.C.S.
Carman, John Ambrose, M.D., B.S. (Lond.), L.R.C.P., M.R C.S.
Coden, Bernard, M.B., Ch.B. (Edin.).
Daplyn, Phyllis Frances Lucy, L.R.C.P., M.R.C.S.
Dawkins, Charles John Massey, M.B., B.Ch. (Camb.), L.R.C.P., M.R.C.S.
Deane, Maslen Mackenzie, M.B., B.S. (Melb.), M.R.C.S.
Galbraith, Margaret Louise Agnes, M.B., Ch.B. (Birm.), L.R.C.P., M.R.C.S.
Grant-Whyte, Harry, M.B., Ch.B. (Cape Town).
Goldman, Victor Abraham, L.R.C.P., M.R.C.S.
Havers, Geoffrey Gordon, L.R.C.P., M.R.C.S.
Hudson, Maurice William Petre, M.B., B.S. (Lond.), L.R.C.P., M.R.C.S.
James, Ian Clinton, M.B., B.S. (Melb.).
Jones, Ivor Davenport, L.R.C.P., M.R.C.S.
Jones, Roland Neville, M.B., Ch.B. (Birm.), L.R.C.P., M.R.C.S.
Kennedy, Agnes Winifred O'Dwyer, L.R.C.P., M.R.C.S.
Mallinson, Frank Barnett, L.R.C.P., M.R.C.S.
Mansfield, Ruth Evelyn, L.R.C.P., M.R.C.S.
Nagle, Patrick Joseph, M.B., B.Ch., N.U.I.
Overton, Percy Michael, M.B., Ch.B. (Leeds).
Picken, Christopher Barugh, M.B., B.S. (Lond.), L.R.C.P., M.R.C.S.
Rice, Raymond Arthur Cracroft, M.B., B.S. (Lond.), L.R.C.P., M.R.C.S.
Woolf, Abraham David, L.R.C.P. & S. (Edin.), L.R.F.P. & S. (Glas.).
Wynter, Tamsin Mary, M.D., B.S. (Lond.), L.R.C.P., M.R.C.S.

We much regret that the names of Messrs. R. E. Apperly and H. P. Crampton were accidentally omitted from our former lists of those who had been granted the D.A. without examination.

ABSTRACTS.

"Anæsthetics for total pneumonectomy." (W. F. REINHOFF, in *Archives of Surgery*, February, 1936, p. 230.

After describing the surgical technique of the operation, the author discusses the question of anæsthetic. He used avertin supplemented by nitrous oxide and oxygen. The anæsthesia is kept very light. Whether or not an intra-tracheal tube should be used is, he thinks, an open question. "It cannot be denied that the insertion of a tube through the mouth into the trachea must always carry organisms into an otherwise sterile region. . . ." Also he maintains that damage to the lining of the trachea would produce a mucopurulent exudation and interfere with the movements of cilia. These factors tend to break down the protective mechanism of the patient. He gives details of a case in which he believes that unskilled passage of the tracheal tube set up damage which, resulting in lobar pneumonia, led to the death of the patient. The simpler the administration of the anæsthetic, the better, is his opinion.

"Post-anæsthetic leucocytosis." (E. M. BOYD, in *Canadian Med. Assoc. Journ.*, February, 1936, p. 159.

Anæsthesia produces in man a leucocytosis independent of the surgical procedure. The leucocytosis reaches a maximum within three to four hours, and is probably due to migration into the blood of cells from liver, spleen, bone marrow, and other tissues. Microscopically, the leucocytosis is indistinguishable from that due to fever or infection, but the chemical composition of the white cells is entirely different from that in fever, late post-operative states, and infection. The effects of anæsthesia on the lipoid composition of the white blood-cells was determined in 19 cases. It was shown that the polymorphoneuclear leucocytes mobilized into the circulating blood after anæsthesia contain decreased amounts of phospholipid, about the same percentage of free

cholesterol, and a medium value for cholesterol esters and neutral fat, as compared with the leucocytes present before anæsthesia.

"Polyneuritis following evipan anæsthesia." (G. B. PALMER, in *New Zealand Med. Journ.*, February, 1936, p. 21.

A woman, 28 years of age, the subject of recurrent melancholia but with no organic nerve defect, was given 0.9 grm. evipan in order that a diagnostic curettage might be performed, as she suffered from menorrhagia. The operation was completed without any unusual symptom. Three days later she complained of poor sight. There was paresis of the right external rectus of the eye, weakness of the left side of the face, especially at the angle of the mouth, and partial paresis of the left side of the palate. After another three days the left external rectus was also paretic. Three months later the paralysis had almost entirely disappeared.

"General anæsthesia with a new ethyl chloride, 'Novanest.'" B. E. ERIGO, P. LINGI and R. GILDO in *Ecora. Ital. di Anest. e. Analges.* December, 1935, p. 505.

The report is based on the use of novanest for 50 operations which included laparotomies, nephrectomies and operations on central nervous systems. The authors find no contra-indication, except broncho-pulmonary lesions or myocardial defect, to the use of this new inhalation anæsthetic which they say acts quickly, producing little excitement, causing no after-effects, and having no deleterious effect on any of the main systems of the body. The agent is given gradually on an Esmarch mask, about 10 c.c. being needed for induction of anæsthesia, after which minute quantities are added at frequent intervals. Recovery follows quickly after the administration stops. The blood-pressure, which rises at first, falls later, but only within narrow limits, and never gave rise to anxiety. The new drug has been prepared by Dr. L. Tirelli, who has conducted extensive chemico-physical researches upon it. The authors do not state its exact composition, but mention ether and chlorinated methylene in its production.

"Physiological and therapeutic action of carbon dioxide inhalation." H. RIVERS and R. B. GONZALES in *Gaceta Med. le Caracas*, November 30th, 1935, p. 339.

The authors regard the introduction of carbon dioxide inhalation into the practice of surgery as one of the most valuable innovations of the day. They give a long account of the action of the gas as illustrated by experiments on animals and in its effects on the human subject, and lay due stress on its importance as a respiratory regulator. The production of acapnia by deficiency of CO_2 is described. They attribute the majority of accidents during inhalation anæsthesia to perturbation of the respiratory centre which is due, in their opinion, either to acapnia or to toxic elements. In this connexion they discuss atelectasis, reflex broncho-constriction, and reflex vasomotor dilatation of the bronchial vascular system, none of which factors can, they believe, be regarded as causing the deaths under discussion. They regard hyperventilation by CO_2 inhalation after operation as the chief means of avoiding post-operative lung complications.

"Making ether an ideal anæsthetic." W. W. KEMP in *Canadian Med. Assoc. Journ.*, April, 1936, p. 409.

It is generally admitted that in the great majority of operations ether suitably given can provide perfect conditions for the surgeon. It is the patient's troubles afterwards that make the anæsthetist chary of employing ether as a routine. These troubles, Kemp believes, can be entirely or largely eliminated by proper pre-operative care and treatment. Based on experimental and clinical grounds his recommendations are: (1) At least three days' stay in hospital before operation, (2) ten minims of Lugol's solution with six grains of desiccated suprarenal cortex daily for five days before operation, (3) for a week before operation a diet which is without meat and has high carbohydrate content and abundance of calcium and vitamins, and 1.5 ounces of lactose daily. Routine removal of mucus at close of operation, by endotracheal suction, is also regarded as one of the important measures in controlling post-operative nausea and sickness.

"Lesions of the nervous system from barbiturism." J. Shermittee in *Anæthésie e Analg.*, February, 1936, p. 1.

Increasing use of the barbiturates by anæsthetists and of suicides from veronal, luminal, etc., have, says the author, drawn attention to the modifications of the nervous system provoked by barbituric intoxication. After massive introduction of barbiturates, when the acute symptoms have passed, there may be left mental troubles accompanied by ocular paralysis pointing to peduncular origin, or a clinical complex resembling that of cerebral tumours or even of disseminated sclerosis. Unfortunately, post-mortem and histological investigations have not equalled the clinical observations, for the former have commonly not fallen into the hands of those qualified to carry them out expertly. Authorities differ as to the region of the encephalon on which falls the brunt of barbituric intoxication. The author concludes that in massive intoxication it is impossible to localize the lesion, the whole central nervous system being subjected to a "brutal toxic submersion," but in subacute intoxications it may be possible that the lesions may carry the mark of a specialized pathological assault, and that this is in the regions most rich in myelinated fibres.

"Epidural anæsthesia." C. Odom in *New Orleans Med. Surg. Journ.*, April, 1936, p. 624.

The author reports highly favourable results in 100 subjects. He reviews the origin of epidural anæsthesia, the anatomical facts involved and the work of Dogliotti. He describes an arrangement whereby it is made easier for the anæsthetist to be certain when his needle has entered the epidural space and that he has not penetrated the dura and gone into the cerebro-spinal fluid. This doubt really constitutes the main difficulty in using epidural analgesia, a method which has obviously great advantages of safety and wide applicability. It can be used for high regions of the body without the risk necessarily run when endothecal analgesia is employed for the same purpose. The anæsthetic employed is two per cent novocain solution and generally 50 c.c. represent the highest amount required.

"Importance of oxygen and of precise dosage of CO_2 *in anæsthesia."* R. MONOD and H. ARUAL in *Anesthe. et Analges.*, April 1936, p. 244.

In a long article advocating certain alterations in the Ombrédanne inhaler and other closed apparatus, the authors discuss the physiology of carbon dioxide in anæsthesia. They quote authorities to show the baneful effects on the liver cells of too high a concentration of the gas, and believe that post-operative vomiting and post-operative mortality in diabetics are attributable to the same cause. In advocating the use of continuous or frequent small amounts of oxygen during anæsthesia, they believe that impoverishment in oxygen more than counterbalances the advantages of semi-closed methods. With regard to the use of CO_2 they state that though it may often be used to hasten the induction it should rarely be introduced in the course of anæsthesia. They are in accord with British authorities in seeking to avoid cyanosis entirely throughout operation.

REVIEW.

"Practical points in anæsthesia." H. K. ASHWORTH, M.B. pp. 160; illustrated. Price 7*s* 6*d*. (J. & R. Churchill, London.)

This is a small volume likely to be of great use to the student who is undergoing practical instruction in the administration of anæsthetics. Although the more complicated methods and apparatus are not dealt with, yet the instructions given regarding those procedures which are most likely to be employed in actual practice by the average practitioner are clearly stated, and with that attention to detail which is so important if practical advice is to be of real use. The Sankey method of induction, which is approved of by the author for routine use, will be new to many readers; it differs in detail from the CE, open E, method which was for years so widely used in several London schools. The author gives a chapter to basal narcosis and one, supplied by W. H. T. Simmons, deals with local methods of analgesia.